SHORT REPORTS

Mortality in sodium chlorate poisoning

Sodium chlorate poisoning is rare but is associated with a high mortality rate,[1] death occurring from massive intravascular haemolysis and acute renal failure. We report the outcome in 14 patients poisoned by sodium chlorate, with special regard to the amount ingested and subsequent management.

Patients, treatment, and results

During 1974-8 we followed up in detail 14 cases of sodium chlorate poisoning referred to the National Poisons Information Service (Guy's Hospital). Data concerning the amount of substance ingested, medical management, and subsequent outcome were obtained (see table). Mortality was high (64%), and death invariably occurred, irrespective of treatment, when the amount of sodium chlorate ingested exceeded 100 g. Early deaths—that is, those within 24 hours—were more common in accidental self-poisoning, when specific antidotes or reducing agents were not administered owing to the delay in diagnosis. Supportive management alone was successful in only one patient (case 12); in this case the ingested amount was less than one-tenth of the stated fatal dose of 20-30 g. In the four other survivors recovery was associated with ingested amounts of 100 g or less of sodium chlorate, prompt administration of sodium thiosulphate or reducing agents, and management of acute renal failure by peritoneal dialysis or haemodialysis. The success of treatment with specific antidotes followed by dialysis is best seen by comparing cases 1 and 14, in which the amounts of sodium chlorate ingested were identical. The clinical features of sodium chlorate poisoning occurred in the following frequency: nausea and vomiting (11 patients; 79%), cyanosis (seven; 50%), abdominal pain (five; 36%), diarrhoea (three; 21%) and dyspnoea (three; 21%). Two patients were admitted in coma and died shortly afterwards. Seven patients became anuric within 48 hours after admission to hospital. Methaemoglobinaemia was found in 13 patients, in two of whom peripheral blood films showed the presence of ghost cells and Heinz-body formation. In the patients who died a constant necropsy finding was a "chocolate" discoloration of the blood and viscera due to staining by bilirubin and methaemoglobin.

Comment

Sodium chlorate is a powerful oxidant used extensively as a herbicide. A white crystalline substance, it is applied dissolved in water. The crystals may be mistaken for sugar with fatal results (cases 3 and 8). Poisoning usually results from ingestion but has been reported after inhalation of atomised droplets.[2] Initial symptoms relate to the irritant effect of the chlorate ion on the gastrointestinal mucosa. After absorption haemoglobin is rapidly oxidised to methaemoglobin and intravascular haemolysis results. Cyanosis becomes clinically detectable when the proportion of methaemoglobin exceeds 10%; values above 70% are fatal. Death occurring within a few hours of ingestion is attributed to tissue hypoxia due to severe methaemoglobinaemia or hyperkalaemia resulting from massive haemolysis. Sodium chlorate is nephrotoxic and causes acute tubular necrosis; the ensuing renal failure may be compounded by haemoglobinuria.

In cases presenting early initial management should comprise gastric lavage and administration of activated charcoal. Sodium thiosulphate (2-5 g in 200 ml of 5% sodium bicarbonate) is a specific antidote that inactivates the chlorate ion and may be given by mouth or intravenously. Methaemoglobinaemia is best treated by giving intravenous methylene blue (20-50 ml of a 1% solution), which is superior to ascorbic acid in reducing methaemoglobin to haemoglobin.[3] Oxygen is of no value. Sodium chlorate is freely dialysable, and early treatment of renal failure by peritoneal dialysis or haemodialysis is recommended. Despite isolated reports of success in treating severe sodium chlorate poisoning,[4 5] the mortality rate remains extremely high. Sodium chlorate is freely available and is not listed as a poison: preparations of it are therefore not required to carry any warning to this effect.

[1] Dérot, M, *et al*, *Semaine des Hôpitaux de Paris*, 1948, **24**, 719.
[2] Jackson, R C, Elder, W J, and McDonnel, H, *Lancet*, 1961, **2**, 1381.
[3] Jaffé, E R, and Newmann, G, *Nature*, 1962, **202**, 607.
[4] Knight, R K, Trounce, J R, and Cameron, J S, *British Medical Journal*, 1967, **3**, 601.
[5] Lee, D B, *et al*, *British Medical Journal*, 1970, **2**, 31.

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Painful gynaecomastia treated with tamoxifen

Unilateral or bilateral gynaecomastia may be a consequence of impaired liver function or lung cancer or occur after certain drugs, notably digoxin, spironolactone, metoclopramide, and cimetidine. In lung cancer gynaecomastia is associated with gonadotrophin-secreting tumours, and there is also an association with hypertrophic pulmonary osteoarthropathy.[1] Histologically the tumour is usually anaplastic large-cell carcinoma, but there are reported cases with squamous-cell carcinoma, adenocarcinoma, and oat-cell carcinoma.[2] Gynaecomastia is usually no more than embarrassing for the patient, but rarely it may be painful. This was the case in the three patients described below. The oestrogen antagonist tamoxifen successfully relieved the pain.

Case reports

Case 1—A 64-year-old heavy smoker presented with weight loss and non-productive cough. Chest radiography showed a right upper lobe opacity with extensive hilar enlargement. Sputum cytology disclosed an oat-cell carcinoma of the bronchus. On presentation he had bilateral gynaecomastia of recent onset. Biochemical liver function values and a liver scan were normal. The gynaecomastia progressed and became intensely painful with little relief from analgesics. Liver function values were abnormal and the serum oestradiol concentration was 455 pmol/l (124 pg/ml) (normal range 55-147 pmol/l; 15-40 pg/ml). He was given tamoxifen 10 mg twice daily. The gynaecomastia regressed and became painless in two weeks. He

Details of 14 cases of sodium chlorate poisoning showing management and outcome

Case No	Age	Sex	Amount ingested	Deliberate or accidental	Initial management	Further management	Outcome
1	55	F	100 g	Deliberate		Supportive	Died (3 hours)
2	55	M	150 g	,,	Methylene blue, ascorbic acid	Mannitol hydrocortisone	Died (36 hours)
3	3	M	Unknown	Accidental		Supportive	Died (6 hours)
4	23	M	100-150 g	Deliberate	Sodium thiosulphate, ascorbic acid	Blood transfusion, hydrocortisone, antibiotics	Died (48 hours)
5		F	30 g	,,	Methylene blue, ascorbic acid	Peritoneal dialysis, blood transfusion	Died* (5 days)
6	25	M	50 g	,,	Methylene blue	Haemodialysis, exchange transfusion	Died†
7	28	M	300 g	,,	Methylene blue	Peritoneal dialysis, exchange transfusion	Died
8	46	F	15 g	Accidental		Supportive	Died (20 hours)
9	48	M	Unknown	Deliberate	Methylene blue, ascorbic acid	Supportive	Died‡ (2 hours)
10		F	45 g	,,	Sodium thiosulphate	Peritoneal dialysis	Recovered
11	18	F	30 g	,,	Sodium thiosulphate, methylene blue	Peritoneal dialysis	Recovered
12	47	M	1-2 g	Accidental	Sodium thiosulphate	Supportive	Recovered
13	13	M	5 g	,,		Peritoneal dialysis, blood transfusion	Recovered
14	18	F	100 g	Deliberate	Methylene blue, sodium bicarbonate	Exchange transfusion, haemodialysis	Recovered

*Necropsy showed portal vein thrombosis. †Fatal cardiac arrest during dialysis. ‡Severe hyperkalaemia present.